Anesthesia Does Not Cause Metabolic Stress

To the Editor:—In their otherwise excellent review, Drs. Blackburn, Maini and Pierce state that: "the induction of anesthesia initiates the (metabolic) response to injury, while most surgical procedures 60 to 90 min in duration do not augment this stress further." We believe that this statement is not correct. First, the documentation given for the above statement is incorrect, since the article referred to deals with the extent and composition of postoperative weight loss. Second, several studies have shown that anesthetic

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agents per se play only a minor role in the observed hormonal changes seen during surgical procedures 60 to 90 min in duration. Existing evidence would suggest that anesthesia is not an important initiator of endocrine-metabolic alterations during or after surgical stress.

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Hypovolemia and Hypotension with Carcinoid Syndrome

To the Editor:—We have read with great interest the case report of the anesthetic care of a patient with carcinoid syndrome by Drs. Patel et al.1 We have had considerable experience anesthetizing patients with carcinoid syndrome2 and would like to call attention to one very important aspect of anesthetic care, namely, the preoperative treatment of dehydration and hypovolemia. Hypotension is the greatest problem for the anesthetist during and after operations on patients with carcinoid syndrome.3,4 Some decrease in blood pressure occurs in all patients owing to the vasodilator effects of virtually all anesthetics. Preoperatively, these patients often have severe diarrhea as well as hypertension, both of which will promote dehydration and extreme hypovolemia. The clinical findings of hypovolemia are poor tissue turgor, slow capillary refill, and a marked decrease in the amount of urine per 24-hour period. Fluctuations in blood pressure may occur during handling of the tumor, but these do not necessarily lead to hypotension when there is a sufficient circulating blood volume, ensured by vigorous preoperative fluid therapy.

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Dose Limits to Acute Nitroprusside Therapy Challenged

To the Editor:—We wish to comment on a letter by Professor Katz.1 Our suggestion for a maximum dose of sodium nitroprusside (SNP) of 1.5 mg/kg2 was made on the basis of reported blood cyanide levels in cases of deliberate cyanide poisoning and SNP overdose. We concluded from these cases that plasma cyanide levels of about 10 μmol/l could be lethal and levels below this might well prove toxic during the inevitably prolonged exposure to cyanide during SNP infusion.3 Indeed, the fact that in vitro the terminal respiratory enzyme cytochrome oxidase is inhibited by 50 per cent by 1.5 μmol cyanide/l makes a lower toxic plasma level very likely. It is probable that there would be a concentration gradient between plasma and tissue, and an upper limit to plasma cyanide levels of 3 μmol/l was suggested. Work in patients demonstrated a correlation between total dose of SNP and plasma cyanide levels, and extrapolation of these results indicated that 3 μmol cyanide/l plasma could result from a total dose of 1.5 mg SNP/kg for hypotensive anesthesia of short duration.2 Our work in dogs, soon to be reported, indicates that this

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